CONDITIONED DIMINUTION OF THE UNCONDITIONED RESPONSE

By

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INTRODUCTION

Recently, researchers in the area of classical conditioning have become increasingly interested in the influence of inhibitory factors in the conditioning process. Inhibitory effects are frequently observed in eyelid and GSR conditioning studies in the form of decrements in the amplitude of both the CR and the UCR during acquisition. With regard to these responses it has been traditional to interpret decreases in their amplitude during reinforcement as due to adaptation. Hilgard and Marquis (1940) define adaptation as "the decrement in a response which is a consequence of its repeated elicitation." In this sense adaptation generally implies reflex fatigue and further suggests that its occurrence signals that some physiological limit has been reached. That adaptation is inadequate as an interpretation of such decrements has become increasingly clear during the last few years.

Grings (1960) has summarized a number of studies which indicate that adaptation cannot account fully for CR decrements during conditioning, in that slight changes in the intensity of either the CS or UCS were sufficient to restore the OR to its previously higher level. It is particularly noteworthy that recovery in response amplitude occurred when stimulus changes were in a downward as well as an upward direction. These results suggest that some sort of central or learned inhibition was operative during the learning process rather than reflex fatigue or adaptation. If this is the case then a logical assumption would be that this inhibition ought to be capable of being disinhibited. Meryman (1953), employing a "probe stimulus" technique, interpolated a soft click occasionally in the inter-stimulus interval during classical GSR conditioning, and found that GSRs to the click were produced and sustained in amplitude in spite of the fact that CR decrement had set in fairly early during acquisition.

Kimmel and Fowler (1961) reported results similar to those of Meryman, using a slightly different GSR conditioning procedure, in which nonreinforced test trials of a compound of the original CS plus a novel stimulus was presented after 4, 8, or 12 regular reinforcement of just the CS. This study also indicated that the extent to which OR inhibition developed varied as a function of the number of reinforced trials. This result also suggests that the inhibitory process may be learned or viewed as a conditioned inhibition, in that it appears to resemble the conditioning process itself, at least with regard to the effect of number of reinforcements.

It would appear that these studies have alluded to an inhibitory process under the control of the CS as possibly responsible for CR decrement rather than such decrement representing an adaptation effect. Furthermore these studies have implications towards reasons why the UCR also tends to become reduced in amplitude during the course of conditioning. If it is assumed that an active inhibitory process under the control of the CS is responsible for UCR as well as CR decrement, it should be possible, on logical grounds, to remove this inhibition by omitting the CS at some point during acquisition.

Recovery of UCR amplitude has in fact been produced in this way by Kimble and Ost (1961) using the eyelid response, and by Kimmel and Pennypacker (1962) using the GSR. Kimble and Ost were successful in eliminating UCR decrement by introducing 10 trials of UCS alone following 50 conditioning trials. In their study the CS was a neon light and the UCS an airpuff. The UCS-only trials produced a significant increase in UCR amplitude over the reduced UCR amplitudes which had been established during acquisition. These investigators also found that the amount of UCR recovery varied as a function of the inter-stimulus interval in the same way that the inter-stimulus interval has been shown to influence the excitatory conditioning process. That is, maximum UCR recovery occurred in those Ss trained with a CS-UCS interval of 0.5 sec. Kimmel and Pennypacker found that amount of recovery of the unconditioned GSR increased as a function of the number of reinforced trials prior to "test" trials on the UCS alone. Taken together these studies lend further support to the contention that conditioned inhibition is one of the factors responsible for the UCR decrement observed in classical conditioning.

In light of the above mentioned experiments, it would seem that the strength of an active inhibitory process under the control of the CS should depend upon whether or not the CS is present throughout the entire CS-UCS interval. That is, more inhibition should be generated in a delayed conditioning paradigm (in which the entire CS-UCS interval is filled with the CS) than in a trace paradigm (in which the CS terminates before the UCS begins). If this is the case, and, if this inhibition is responsible for the attenuation of the UCR, then the UCR should be attenuated more in a delayed than a trace procedure. Furthermore any recovery of UCR amplitude that can be demonstrated on trials containing only the UCS should occur to a greater degree in the delayed than in the trace condition.

The purpose of the present study was to evaluate empirically these deductions. Since it has been shown that the CS appears to play the crucial role in UCR decrement it was decided to perform an experiment in which the effects of CS duration on the UCR diminution phenomenon could be evaluated. Two specific hypotheses were evaluated:

1. That differences in CS duration (i.e. Delayed vs. Trace) would produce differential diminution of the UCR (in favor of Delayed) in classical GSR conditioning;
2. That amount of UCR recovery on UCS-only test trials would vary (in favor of Delayed) as a function of the CS duration (Delayed vs. Trace) which obtained during acquisition in classical GSR conditioning.

METHOD

Subjects

Sixty-four[1] undergraduate students at the University of Florida volunteered to serve as Ss. The sample contained 39 males and 25 females. The Ss were assigned randomly to the experimental conditions.

There were 32 experimental (paired CS-UCS) and 32 control (unpaired CS and UCS) Ss. The 32 experimental Ss (20 men and 12 women) contained two subgroups of 16 Ss each, one subgroup for which the CS duration was 1.0 sec. (Trace group) and the other for which the CS duration was 5.0 sec. (Delayed group). The 32 control Ss (19 men and 13 women) also contained two subgroups of 16 each, one for which the CS was 1.0 sec. duration and the other for which it was 5.0 sec.

Instructions

All instructions were read to Ss by E. These appear in Appendix A. The instructions informed the Ss about the general nature of the experiment and the galvanic skin response (GSR). The stimuli to be used were mentioned and it was indicated that this was an experiment on the effects of different stimuli on the GSR. Each S was told to pay close attention to the stimuli and to refrain from making any movements other than those minimally necessary to remain comfortable.

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[1] Five Ss had to be replaced, three because of equipment failure and two because of E's errors.

Apparatus

Specification of the CS

The CS was a 1,000 cps tone of either 1.0 or 5.0 sec. duration. Its intensity was 40 db. (re: .0002 dynes/cm^2, rated at the earphones). The tones were produced by General Radio Company equipment and delivered via Trimm ANB-1 headphones with MX-41-AR cushions.

Specification of the UCS

The UCS was an electric shock of 0.1 sec. duration, produced by a Psychological Instruments Company stimulator, and delivered via silver-coated electrodes to the index and middle finger of S's right hand. Its intensity (open circuit) was 120 v.

Specification of stimulus durations and temporal relations

Two Hunter silent interval timers were used to deliver the CS of either 1.0 or 5.0 sec. duration and to deliver the UCS, 5.0 sec. after the CS's onset, on paired CS-UCS trials only. On unpaired CS-UCS trials the UCS occurred 30-60 sec. following the CS's onset. For all

Ss the inter-trial (a "trial" consisting of one CS and one UCS) interval was 30-60 sec.

Presentations of the CS without shock (during adaptation trials) were separated by intervals of 20-30 sec. Between the preliminary trials of the UCS alone there was also a 20-30 sec. interval. On the test trials involving only UCS presentations the inter-trial interval was again 20-30 sec.

Measurement of the GSR

The GSR was picked up by a NaCl jelly-metal skin junction employing 3/4 in. lucite cups which contained zinc electrodes. The electrodes were attached to the back and palm of the S's left hand by adjustable rubber straps approximately 5/8 in. wide. Alcohol was used to clean the surface of the skin before the GSR electrodes were attached. The zinc electrodes were covered with a few drops of zinc sulphate solution prior to the cup being filled with the NaCl jelly.

The GSR was measured as the maximum drop in skin resistance which occurred within 4.0 sec. after the onset of a stimulus.[2] After being picked up from the S's left hand, the response was amplified (Hunter GSR amplifier) and recorded on an Esterline-Angus ink-writing milliammeter with a paper speed of 3 in./min.

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[2] When the stimulus was the CS, this response was an "anticipatory" CR. When the stimulus was the UCS, this was a UCR.

For statistical purposes all responses were converted to the square root of the change in conductance ($\sqrt{\Delta C}$).

Procedure

After _E_ finished reading the instructions to _S_, he prepared and attached the GSR electrodes to _S_'s hand. _S_ was then seated in a reclining chair in a sound-insulated, air-conditioned room illuminated by a 7.5 w. semi-shielded bulb. The GSR and UCS electrodes were then appropriately attached. Finally, the earphones were placed on _S_ and comfortably adjusted. During this procedure _S_s were briefly reminded about the function of each piece of apparatus.

When the equipment had been attached, _E_ closed the door to the experimental room and returned to the adjoining room which houses the equipment. Five min. were allowed to elapse for _E_ to adjust and calibrate the apparatus and for _S_ to adapt to the experimental room. Then _S_ received 3 preliminary trials of the UCS alone. On these trials the UCS intensity was increased in steps until, on the third trial, the UCS intensity to be used thereafter was reached. Following these 3 shock trials, the CS was delivered alone for 10 trials, or for as many trials as were necessary to produce 2 consecutive zero deflections on the Esterline-Angus milliammeter, whichever came first. No fewer than 4 CS-only trials were given to any S.

Each S was then given 16 acquisition trials of paired or unpaired CS-UCS presentations according to the condition to which he had been assigned. Following these 16 acquisition trials, each S then received 4 test trials on the UCS alone, with the same inter-trial interval as during training. Because of the UCS-only test trials following conditioning, it was not possible to include an extinction phase.

The CS adaptation series for the Trace groups was carried out with the 1.0 sec. tone and for the Delayed groups with the 5.0 sec. tone. For the 16 acquisition trials in the Trace-Paired condition the UCS occurred 5.0 sec. following the CS's onset. For the 16 acquisition trials of Ss in the Delayed-Paired condition the UCS also occurred 5.0 sec. after the CS's onset. For Ss in the Unpaired conditions the UCS followed the CS's onset by 30-60 sec. Thus, the Ss in the two control conditions received the same number of stimulus presentations as did their experimental counterparts, in the same overall period of time. The only difference was that in the former group there were no paired CS-UCS presentations while in the latter there were.

When the 4 test trials of UCS-only were completed each S was thanked for his participation, asked not to discuss the experiment with others, and then dismissed. The entire procedure for collecting data from each S required between 30-40 min.

RESULTS

Description of the response units

Kimmel has shown (1959) that the square root of the change in conductance provides a GSR unit which is appropriately distributed and has sufficient reliability for statistical analyses.

Tests of assumptions and hypotheses relative to acquisition

To evaluate the main experimental hypotheses regarding UCR decrement, it was necessary first to provide support for the assumption that conditioning had occurred. It has been shown (Kimmel, 1964) that groups receiving unpaired CS-UCS presentations provide adequate controls for a test of the conditioning assumption. The dependent variable used to examine this question was the GSR magnitude, in response to the CS, of all Ss on each of the 16 acquisition trials for the experimental (Paired) and control (Unpaired) groups, ignoring the CS-duration variable.

Table 1 presents a summary of the analysis of variance of the acquisition CR magnitudes.

Fig. 1--Mean CR magnitudes during acquisition for paired and unpaired groups.

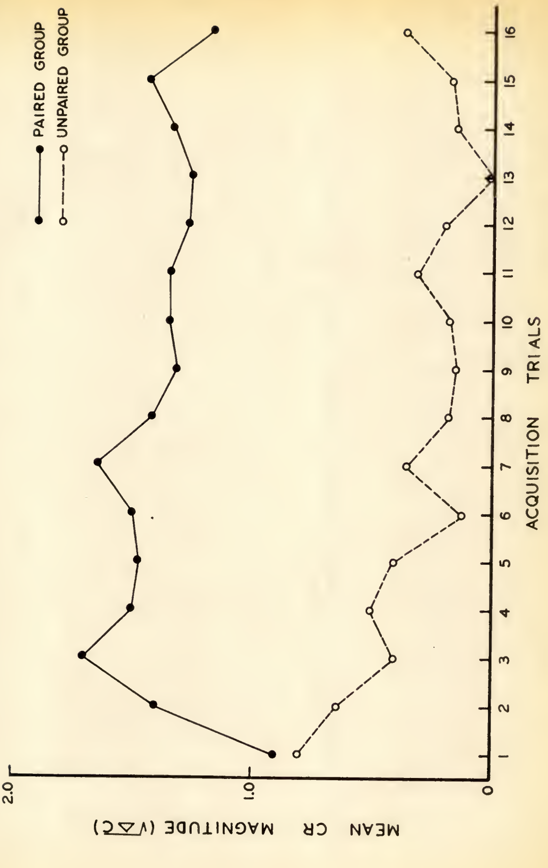
PAIRED GROUP
UNPAIRED GROUP
MEAN CR MAGNITUDE (√ΔC)
2.0
1.0
0
1
2
3
4
5
6
7
8
9
10
11
12
13
14
15
16
ACQUISITION TRIALS

TABLE 1

Analysis of Variance of Acquisition CRs

Source	df	MS	F
Trace-Delayed (CS-duration)	1	15.31	-
Pairing	1	291.81	50.05***
Trace-Delayed x Pairing	1	30.67	5.26*
Error (between)	60	5.83	-
Trials	15	.93	2.45**
Trials x Trace-Delayed	15	.57	-
Trials x Pairing	15	1.52	4.00**
Trials x Trace-Delayed x Pairing	15	.35	-
Error (within)	900	.38	-

*P < .05
**P < .01
***P < .001

The analysis summarized in Table 1 showed that the overall difference in average CR magnitude during acquisition between Paired and Unpaired groups was highly significant ($p < .001$). The effect of Trials was also significant ($p < .01$) as were interactions of CS-duration x Pairing and Trials x Pairing ($p < .05$ and $p < .01$ respectively). Thus it can be seen that Pairing was associated with greater CR magnitudes during acquisition (and, incidentally, that this effect was greater for the Delayed than for the Trace groups). Also, it should be noted that the Paired and Unpaired groups changed differently over the 16 acquisition trials, the former increasing while the latter decreased.

Since these tests supported the assumption that conditioning occurred, it was appropriate to evaluate the results relating to the main experimental hypotheses. The dependent variable used to evaluate this phase of the study was the magnitude of the GSR to the UCS on each of the 16 acquisition trials. Figs. 2 and 3 show the average values of this measure on each acquisition trial for the two paired and the two unpaired groups.

It can be seen in these figures that, while all four groups showed some reduction in average UCR magnitude during acquisition, the Paired groups' UCRs dropped well below the level reached by the UCRs of the Unpaired groups. The figures also show that, of the two Paired groups, the UCRs of the Delayed-CS group were lower than those of the Trace-CS group, while, in the Unpaired groups, this difference was reversed.

Table 2 presents a summary of the analysis of variance of the acquisition UCRs.

The analysis summarized in Table 2 showed that the overall difference in UCR magnitude during acquisition between the combined experimental and control groups was significant ($p < .05$). Differences due to Trials were significant ($p < .01$), since there was diminution of the UCR over trials in all four groups. The Trials x Pairing interaction was significant ($p < .01$), reflecting the fact that the UCR magnitudes in the experimental and the

Fig. 2--Mean UCR magnitudes during acquisition for delayed groups.

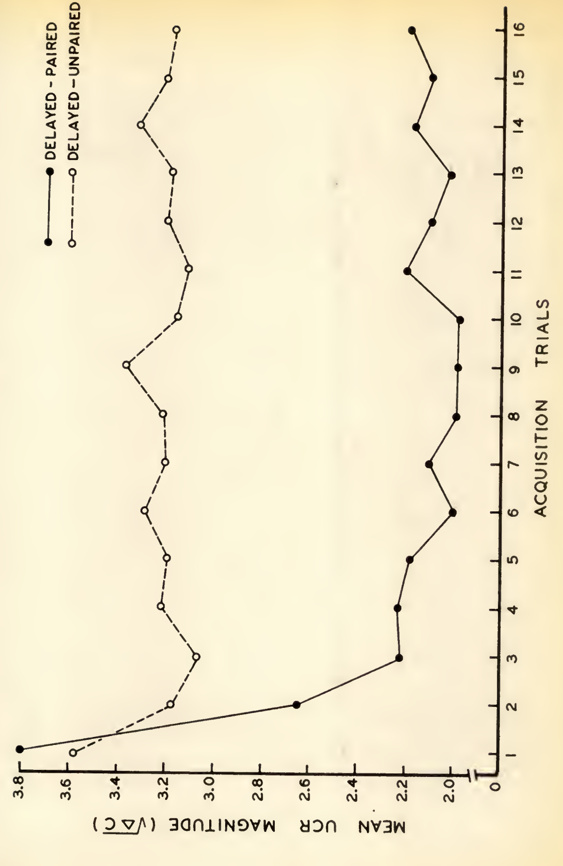
DELAYED - PAIRED
DELAYED - UNPAIRED
MEAN UCR MAGNITUDE (√ΔC)
3.8
3.6
3.4
3.2
3.0
2.8
2.6
2.4
2.2
2.0
0
ACQUISITION TRIALS
1
2
3
4
5
6
7
8
9
10
11
12
13
14
15
16

Fig. 3--Mean UCR magnitudes during acquisition for trace groups.

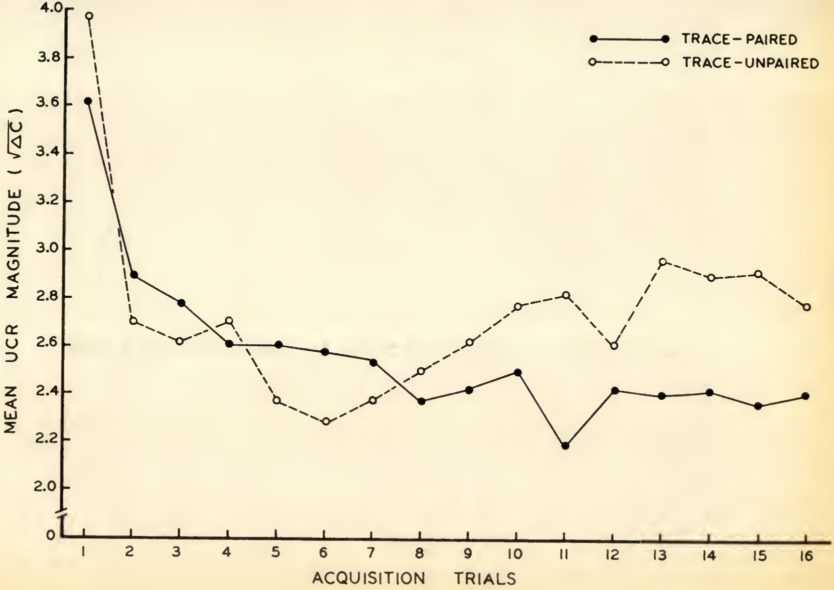
TRACE - PAIRED
TRACE - UNPAIRED
MEAN UCR MAGNITUDE ($\sqrt{\Delta C}$)
4.0
3.8
3.6
3.4
3.2
3.0
2.8
2.6
2.4
2.2
2.0
0
1
2
3
4
5
6
7
8
9
10
11
12
13
14
15
16
ACQUISITION TRIALS

TABLE 2

Analysis of Variance of Acquisition UCRs

Source	df	MS	F
Trace-Delayed (CS-duration)	1	1.46	-
Pairing	1	82.24	5.90*
Trace-Delayed x Pairing	1	42.66	-
Error (between)	60	13.94	-
Trials	15	5.40	16.88**
Trials x Trace-Delayed	15	.39	-
Trials x Pairing	15	.95	2.97**
Trials x Trace-Delayed x Pairing	15	.83	2.59**
Error (within)	900	.32	-

*$P < .05$
**$P < .01$

control groups diminished at different rates over trials. This also is in conformity with earlier results and with the present predictions. The triple interaction (Trials x CS-duration x Pairing) was also significant ($p < .01$), inasmuch as the Trials x Pairing interaction was different in the Delayed and the Trace conditions. In other words there was differential diminution of the UCR over trials between the Paired and Unpaired groups, but this effect was not as great in the Trace as in the Delayed conditions. This, of course, also supports the first experimental hypothesis.

Table 3 presents a summary of a separate analysis of variance of the UCR magnitudes on the 16 acquisition trials for the 32 Ss in the Trace groups.

TABLE 3

Analysis of Variance of UCRs for Ss in Trace Groups

Source	df	MS	F
Pairing	1	3.23	-
Error (between)	30	15.47	-
Trials	15	3.38	13.55***
Trials x Pairing	15	.70	2.80**
Error (within)	450	.25	

**P < .01
***P < .001

The analysis summarized in Table 3 showed that the overall mean difference in UCR magnitude during acquisition between the Trace-experimental and the Trace-control groups was not significant. The Trials effect and the Trials x Pairing interaction were significant (p .001 and p .01 respectively) indicating that there was a differential rate of diminution of the UCR over trials and that this rate was more pronounced in the Paired group.

Table 4 presents a summary of analysis of variance of UCR magnitudes on the 16 acquisition trials for the 32 Ss in the Delayed groups.

TABLE 4

Analysis of Variance of UCRs for Ss in Delayed Groups

Source	df	MS	F
Pairing	1	121.61	9.81**
Error (between)	30	12.40	-
Trials	15	2.22	5.69***
Trials x Pairing	15	1.27	3.26***
Error (within)	450	.39	-

**P < .01 ***P < .001

The analysis summarized in Table 4 showed that the mean difference in UCR magnitude during acquisition between the Paired-Delayed and the Unpaired-Delayed group was highly significant ($p < .01$), indicating that there was greater UCR decrement under the Delayed-Paired condition than under the Delayed-Unpaired (control) one. (It is recalled that this difference was not significant in the Trace condition). Both the Trials effect and the Trials x Pairing interaction were also significant ($p < .001$), indicating that there was a differential rate of diminution of the UCR over trials between the two subgroups. Actually, as is shown in Fig. 2, the Delayed-Unpaired group showed hardly any UCR reduction at all while the Delayed-Paired group's UCRs dropped lower than any of the others.

Tests of assumptions and further predictions relative to UCR decrement and recovery

Since the first experimental hypothesis regarding UCR decrement was supported empirically it was reasonable to assume that the phenomenon would also be reflected in:

1. UCR acquisition magnitudes which represented a significantly greater decrement from UCR adaptation magnitudes in the conditioning groups than in the control groups;
2. UCR test trial magnitudes in the conditioning groups which would show greater recovery

than the UCR test trial magnitudes in the control groups (Hypothesis No. 2); and

3. A greater degree of UCR recovery on test trials in the Delayed-Paired group than in the Trace-Paired group, but no analogous difference in a comparison of the Delayed and Trace-*Unpaired* subgroups.

UCR magnitude difference measures

To evaluate the first of these predictions the average UCR adaptation magnitude (*A*) was computed for each *S*. Since the degree of UCR reduction has been shown to vary positively with number of reinforcements (Kimmel and Pennypacker, 1962), it was decided to use the average UCR magnitude on the last 4 acquisition trials (*B*) for each *S* as the acquisition UCR value. Then, *B* was subtracted from *A* to arrive at a UCR magnitude difference measure for each *S*. Appendix B shows these difference measures for all *S*s in each of the experimental groups.

Table 5 presents a summary of the analysis of variance of the UCR difference measures (*A*-*B*).

The analysis summarized in Table 5 showed that the mean UCR decrement difference (*A*-*B*) between conditioning and unpaired control groups was significant ($p < .01$). Thus, it can be seen that the Pairing effect which

TABLE 5

Analysis of Variance of UCR Difference Measures

Source	df	MS	F
Trace-Delayed (CS-duration)	1	.61	-
Pairing	1	9.40	11.60**
Trace-Delayed x Pairing	1	.99	-
Error (within)	60	.81	-

**$P < .01$

obtained during acquisition resulted in a significantly greater departure of the UCR during acquisition from the pre-acquisition level in the Paired groups than in the control groups.

The second prediction was evaluated by computing the average UCR magnitude on the 4 test trials (*C*) for each *S*, and subtracting from this value each *S*'s average UCR magnitude on the last 4 acquisition trials (*B*). Appendix B presents these increment difference measures (*C*-*B*) for all *S*s in each of the experimental groups.

Table 6 presents a summary of the analysis of variance of the UCR increment difference measures (*C*-*B*).

The analysis summarized in Table 6 showed that the mean *C*-*B* difference between Paired and Unpaired groups was significant ($p < .01$), indicating that *S*s in the Paired conditions showed a significantly greater degree of UCR

TABLE 6

Analysis of Variance of UCR Increments (C-B)

Source	df	MS	F
Trace-Delayed (CS-duration)	1	.51	2.22
Pairing	1	2.00	8.70**
Trace-Delayed x Pairing	1	.38	-
Error (within)	60	.23	-

$^{**}p < .01$

recovery on the test trials than did those in the Unpaired conditions. The expected interaction was not significant.

To evaluate further the prediction that Ss in the Delayed-Paired group would show a greater degree of UCR recovery on test trials than would Ss in the Trace-Paired group, but that Ss in the two control groups would not differ in this respect, two separate t tests were performed. Table 7 presents the overall means and SDs of the UCR magnitudes and increment difference measures on the last 4 acquisition trials (B), and on the 4 test trials (C) for each of the 4 experimental groups. A t test of the difference between the C-B means of the Delayed-Paired and the Trace-Paired groups was significant ($t = 1.94$ with 30 df, $p < .05$) indicating that more UCR-recovery occurred with the Delayed CS than with the Trace CS. A t test of the difference between the C-B means of the Delayed-Unpaired and the Trace-Unpaired groups was not significant (*t* = .30 with 30 df).

TABLE 7

Means and SDs of UCR Magnitudes and Difference Measures

		B	C	
Conditions	Measure	Last 4 Trials	Test Trials	C-B
Trace-Paired	$\bar{X}$	2.42	2.81	.39
	SD	1.12	.99	.67
Delayed-Paired	$\bar{X}$	2.12	2.78	.72
	SD	1.01	1.04	.49
Trace-Unpaired	X	2.86	3.05	.19
	SD	1.07	1.19	.26
Delayed-Unpaired	$\bar{X}$	3.21	3.43	.22
	SD	1.25	1.11	.39

DISCUSSION

The purpose of this study was to investigate the UCR decrement phenomenon which has been observed in classical conditioning studies, particularly in relation to the duration of the CS during training. Both experimental hypotheses and allied assumptions received support. The hypotheses were:

1. That differences in CS duration would produce differential diminution of the UCR in classical conditioning.
2. That amount of UCR recovery, on UCS-only test trials, would vary as a function of the CS duration which obtained during acquisition.

Since previous research had suggested that the UCR decrement phenomenon was due to conditioned inhibition, it was important to check the assumption that a genuine associative phenomenon was responsible for the decrement. It is the temporal contiguity of the CS and the UCS which becomes the crucial basis for the inference that associative or conditioned inhibition has occurred. The inclusion of the two unpaired control groups in this study served to substantiate the above assumption. Not only did the two unpaired groups serve as controls but they also provided a comparison whereby different degrees in amount of UCR reduction could be evaluated.

As was shown in Figs. 2 and 3, the UCR decrement effect is clearly associated with those Ss who received paired CS-UCS presentations during acquisition. While the overall significance of this effect has been demonstrated between the experimental and control groups it was also expected that such difference would show itself in comparisons of the Delayed-Paired vs. Delayed-Unpaired and Trace-Paired vs. Trace-Unpaired groups. Analysis of variance of the UCRs in these separate conditions showed that the overall UCR decrement was significantly different only in the Delayed-Paired vs. Delayed-Unpaired comparison. The difference in overall mean UCR magnitude during acquisition between the Trace-experimental and the Trace-control groups failed to meet significance. However, that the decrement effect, as a differential diminution in UCR magnitude, also occurred in these groups is quite clear as can be seen in Fig. 3 and also from the fact that the Trials effect and the Trials x Pairing interaction were both highly significant. Diminution of the UCR over trials was more pronounced in the Trace-Paired group than in the Trace-Unpaired group.

Why, then, did UCR decrement not show up as a significant overall effect between the Trace-Paired and Trace-Unpaired groups? Apparently the inhibition phenomenon follows the same course as does the conditioning of emotional reactions generally; more specifically the development of conditioned fear reactions. Mowrer and Aiken (1954) have demonstrated that fear learning is greatest when the CS is contiguous with UCS onset, i.e. a delayed conditioning paradigm is more effective in producing conditioned fear than a trace paradigm. Early in conditioning the Trace CS does not have as aversive an effect as would be necessary to produce a significant overall difference between the Trace-Paired and Trace-Unpaired groups. That the Pairing effect in these two groups is responsible for conditioned inhibition of the UCR can be noted in the fact that the two groups differed significantly in rate of UCR diminution over trials. This finding lends some support to the assertion that the diminution effect is due to a conditioning phenomenon in that it occurs as a developing process similar to the more conventional characteristics of conditioning.

In classical conditioning the CS becomes a cue for or a producer of fear, since it has been associated with the coming of a noxious stimulus such as shock. Because of the temporal relationships between the CS and the UCS, the CS comes to have fear producing properties and is responded to appropriately. But, since the noxious UCS comes, in spite of the CR, this emotional reaction is inefficient and the S learns to inhibit it (Kimmel, 1963). This inhibition represents an economy of behavior. Such inhibition occurs throughout the conditioning process but it is in the later stages of learning where inhibition is greatest that UCR decrement also is observed.

Based on this reasoning, it was expected that a delayed paradigm would result in more inhibition than a trace paradigm, in that the CS is present longer in the delayed condition and inhibition should be greater in the presence of the actual inhibitor than in the presence of traces of it. It seems logical to assume that there would be less initial inhibition to aversive traces. This explanation would seem to be appropriate in evaluating the overall comparison between the Trace-Paired and the Trace-Unpaired groups. That is, whatever inhibitory influences are present in the Trace-Paired group early in conditioning are not sufficiently strong to enable a differentiation to be made between these two groups. However, as the conditioning process becomes established in the Trace-Paired group, the inhibitory effects become quite clear as a progressive phenomenon which ultimately differentiates between the two groups. There is a suggestion in this comparison between the Trace-Paired and the Trace-Unpaired groups that the UCR decrement effect is a characteristic of this type of learning and provides even more support to the notion that conditioned or associative inhibition underlies the UCR diminution phenomenon.

The fear learning study by Mowrer and Aiken is, of course, relevant also to the fact that the Pairing effect resulted in significantly greater CR magnitudes during acquisition in the Delayed-Paired than in the Trace-Paired group. This would mean that fear learning occurred to a lesser degree in the latter group, which fits in with the assumption that less general inhibition was a part of the conditioning process for Ss in the Trace-Paired condition. Also, this finding helps to substantiate the position of previous investigators regarding the decrement or inhibitory effect as occurring in much the same way that the conditioning process occurs.

The second hypothesis, concerning differential diminution of the UCR in the Delayed and Trace conditions, was borne out on two counts. First, the analysis of variance showed a significant difference in rate of UCR diminution between the Paired and the Unpaired groups and, second, the analysis indicated that this effect was more pronounced in the Delayed-Paired than in the Trace-Paired group. Since the only differences between these groups during acquisition were the CS durations and CS-UCS pairing, it can again be seen that the decrement phenomenon is a result of the conditioning process and also, more specifically, that it is dependent upon the presence of the previously paired CS. In this context it would be appropriate for future investigation of this phenomenon to evaluate the effects of CS intensity on UCR decrement, with the prediction, if it is a conditioning phenomenon, that CS intensity would have the same general influence on the inhibitory process as it has on conditioning (Razran, 1957).

Further support for a conditioned inhibition interpretation of the UCR decrement is gained by looking at the data on UCR recovery which occurred on test trials of UCS presentations only. The difference in amount of recovery between the Paired and Unpaired groups was highly significant in favor of the former group.

The most crucial test of whether or not the UCR decrement was due to inhibition under the control of the CS was a comparison of the Trace-Paired and the Delayed-Paired groups on the UCS-only test trials. A *t* test of this difference was significant, providing support for the conclusion that differences in CS duration during acquisition produced more diminution or more inhibition in the Delayed-Paired group than in the Trace-Paired (there was no significant difference in amount of recovery between the Delayed-Unpaired and the Trace-Unpaired groups). This latter finding again points to the fact that diminution of the UCR is a manifestation of an associative process and not attributable to adaptation or reflex fatigue, and, furthermore, that the CS is in control of the inhibitory process.

SUMMARY

The purpose of the present study was to evaluate empirically the UCR diminution phenomenon which has been observed in classical conditioning. Since it had been shown that the CS appears to play the crucial role in UCR decrement it was decided to perform an experiment in which the effects of CS duration on the UCR diminution phenomenon could be evaluated. Two specific hypotheses were considered:

1. That differences in CS duration (i.e. Delayed vs. Trace) would produce differential diminution of the UCR (in favor of Delayed) in classical GSR conditioning;
2. That amount of UCR recovery on UCS-only test trials would vary (in favor of Delayed) as a function of the CS duration (Delayed vs. Trace) which obtained during acquisition in classical GSR conditioning.

An analysis of the acquisition data showed that differences in CS duration during acquisition resulted in a significantly greater UCR diminution effect in the Delayed-CS group than in the Trace-CS group, providing support for the first hypothesis. Furthermore, an analysis of the UCS-only test trial data was also significant, indicating that UCR recovery occurred to a significantly greater degree in the Delayed-CS condition than in the Trace. Analyses of data, with respect to appropriate control groups were not significant, providing support for the contention that the UCR diminution phenomenon is associated with the conditioning process.

In light of the above findings, a conclusion was reached that the attenuation of the UCR which has been observed in classical conditioning is under the influence of the CS. Since the CS, in its temporal relationship to the UCS, appears to be a major determinant of such attenuation it was further concluded that a form of conditioned inhibition is one of the factors responsible for UCR diminution during acquisition.

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APPENDICES

APPENDIX A

INSTRUCTIONS

The experiment in which you are about to participate is a study of the effects of external stimulation on the galvanic skin response. Two kinds of stimuli will be used: tones played through earphones and a mild electric shock to the fingertips of your right hand. All you have to do is pay close attention to the stimuli you will be receiving. The galvanic skin response will be picked up automatically by two contacts attached to the palm and back of your left hand.

The galvanic skin response occurs automatically when certain kinds of stimuli are applied. You are not able to control it nor are you able to feel it when it occurs. It is recorded as a very small change in the electrical resistance of your skin. This change is amplified and recorded on a moving paper record. After the experiment you will be allowed to look at the record of your galvanic skin response.

Remember, your task in this experiment is to pay close attention to the stimuli you will be receiving.

Please do not move around unnecessarily. These things are very important otherwise it will be impossible to determine the effects of the external stimulation on your galvanic skin response. If you have any questions, please ask them now because we will not be permitted to talk during the experiment.

After the earphones are put on and the contacts hooked up, you are to relax and do nothing but pay attention to the stimuli. Try not to move your body or hands and arms any more than is necessary to remain comfortable. Because of the comfortable chair you will be sitting in, you may have a tendency to become drowsy and you may have to remind yourself to pay attention from time to time.

The experiment will take about 30 to 40 minutes. Now the contacts that will pick up your galvanic skin response will be attached to your left hand. Do you have any other questions?

APPENDIX B

SUPPLEMENTARY DATA

TABLE 8

Mean CR Magnitudes ($\sqrt{\Delta C}$) for All 64 Ss on Acquisition Trials

Trials	Trace Paired	Delayed Paired	Trace Unpaired	Delayed Unpaired
1	.91	.90	.96	.63
2	1.17	1.63	.92	.36
3	1.24	2.15	.35	.46
4	1.21	1.79	.47	.53
5	1.07	1.89	.44	.37
6	1.12	1.89	0	.24
7	1.42	1.86	.47	.24
8	1.08	1.75	.38	0
9	.84	1.79	.27	.04
10	1.06	1.66	.27	.11
11	1.11	1.60	.28	.34
12	.90	1.66	.21	.19
13	.87	1.67	0	0
14	1.10	1.56	.30	0
15	1.18	1.69	.22	.15
16	1.06	1.30	.26	.52
Overall Means	1.08	1.67	.36	.26

TABLE 9

Acquisition UCR Magnitudes ($\sqrt{\Delta C}$) for Delayed-Paired Group

S	TRIALS															
	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16
1	4.42	2.46	2.14	1.89	1.91	1.98	1.54	1.87	2.07	1.53	3.14	2.18	1.54	2.41	2.46	2.50
2	3.11	2.12	1.42	2.04	1.74	2.04	2.14	2.14	2.32	2.00	2.18	2.24	2.93	2.59	2.42	2.74
3	4.32	3.30	2.36	1.76	1.16	2.18	2.74	.85	2.04	2.35	2.72	3.10	3.10	2.74	2.83	3.46
4	3.99	3.89	3.69	2.73	2.67	2.73	3.62	2.22	2.10	2.12	2.10	2.57	2.15	2.69	1.24	1.96
5	5.41	2.57	2.86	2.54	2.33	2.07	1.97	1.82	1.99	2.03	1.55	1.87	1.87	1.87	1.66	2.64
6	2.94	3.53	2.27	1.74	1.93	1.68	2.23	2.12	2.07	2.30	2.40	1.99	2.27	2.36	2.35	2.17
7	2.57	3.18	2.35	2.32	2.15	2.50	2.93	2.87	2.89	2.62	2.62	2.71	2.75	2.89	2.93	2.41
8	5.72	2.63	1.89	1.76	1.60	1.71	1.23	.83	1.09	1.20	1.18	.97	1.06	1.24	1.30	1.60
9	3.99	2.70	2.81	2.80	2.97	1.35	2.90	3.01	2.97	1.52	3.60	2.70	1.76	1.82	1.58	.73
10	5.28	2.96	2.44	3.00	3.16	3.32	2.55	3.58	2.49	3.38	3.65	4.00	3.85	4.10	4.03	5.09
11	2.49	0	0	2.95	1.17	1.13	1.12	1.10	0	0	.79	.82	0	.86	.87	0
12	3.26	3.36	2.03	.81	.85	1.10	.73	1.36	1.05	1.21	1.01	1.31	1.26	1.28	1.41	1.52
13	1.76	.79	1.33	1.32	1.71	1.06	1.30	1.48	.75	1.28	1.50	1.28	.73	1.03	1.51	.75
14	3.55	3.02	2.52	2.66	2.38	2.63	2.57	2.92	2.91	2.70	1.62	2.64	2.77	2.09	3.09	3.66
15	1.73	1.40	.97	1.04	1.02	1.05	.96	.93	1.00	1.02	1.05	.94	1.04	1.06	1.12	1.09
16	6.19	5.43	4.58	4.99	5.46	3.16	2.96	2.48	2.62	3.65	3.44	3.26	3.65	3.14	2.48	2.75

TABLE 10

Acquisition UCR Magnitudes ($\sqrt{\Delta C}$) for Trace-Paired Group

S	TRIALS															
	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16
1	2.32	2.08	1.98	1.98	1.94	2.12	1.95	1.57	1.54	1.64	1.19	1.62	1.35	1.32	1.08	1.19
2	3.46	4.04	4.07	3.64	3.69	3.14	3.14	3.78	3.56	3.20	3.51	3.55	3.40	3.58	2.11	3.46
3	2.92	2.89	3.13	2.87	3.52	3.27	3.30	3.38	3.21	3.46	2.92	3.32	3.64	2.99	2.98	3.52
4	5.83	5.65	5.24	5.83	5.07	4.88	5.04	4.12	5.29	5.30	5.41	4.82	5.51	5.54	5.90	5.24
5	5.68	4.22	4.34	3.77	3.33	3.61	3.70	3.77	3.70	3.88	3.32	4.20	3.78	3.94	2.85	3.76
6	4.23	2.09	1.23	1.69	1.88	2.16	2.41	1.50	1.28	2.83	1.57	1.70	2.35	2.76	3.15	2.53
7	4.37	3.00	3.24	2.85	2.90	2.74	1.54	2.26	2.48	3.16	1.81	2.27	2.01	2.50	2.76	1.17
8	2.22	1.37	1.15	1.14	1.14	1.13	.94	.66	1.14	.64	1.55	1.33	1.39	1.39	.67	1.28
9	4.85	2.26	1.93	1.73	1.36	1.50	1.66	1.66	1.53	1.60	1.16	1.95	1.26	1.84	1.56	1.87
10	2.68	3.38	2.91	2.13	2.58	2.67	1.89	1.57	2.18	2.49	1.59	1.84	2.12	1.90	2.37	1.71
11	3.03	1.99	2.36	2.09	1.82	2.05	1.99	1.86	2.04	1.83	2.20	1.97	1.72	1.74	1.55	1.88
12	2.47	2.45	2.46	2.29	2.51	2.47	2.24	2.10	2.24	2.15	2.18	2.30	2.30	1.98	2.03	2.34
13	4.54	3.26	2.44	2.41	3.16	1.60	1.92	1.93	2.14	.88	1.08	1.12	1.42	2.11	2.26	3.00
14	3.23	1.88	.99	1.62	1.74	1.55	1.55	1.48	1.88	1.59	1.81	1.67	1.62	1.36	1.40	1.40
15	2.66	3.38	3.85	3.58	3.70	3.86	3.77	3.68	3.41	3.16	2.96	3.35	3.22	3.14	3.25	3.17
16	3.60	2.07	2.26	2.64	1.78	1.89	2.60	1.96	2.00	1.86	1.83	1.39	1.20	1.82	1.41	1.46

TABLE 11

Acquisition UCR Magnitudes ($\sqrt{\Delta C}$) for Delayed-Unpaired Group

S	TRIALS															
	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16
1	2.31	2.74	3.41	3.83	4.48	4.61	4.63	4.76	4.71	2.75	4.10	3.86	3.29	3.65	4.26	4.02
2	2.92	4.21	3.48	3.91	4.20	4.38	4.05	1.90	3.48	3.68	4.77	2.75	3.79	3.97	3.25	3.30
3	3.13	3.24	2.87	2.81	3.32	3.16	3.21	2.99	3.05	2.97	2.76	2.71	2.97	2.91	2.46	2.51
4	3.18	3.95	3.59	3.85	3.71	3.87	3.60	3.52	3.42	3.91	3.84	3.60	4.31	4.20	4.38	4.29
5	2.10	3.12	3.24	3.46	3.59	3.44	3.69	3.73	4.03	2.87	3.40	3.94	3.81	3.18	1.44	1.98
6	4.14	5.35	4.70	5.35	4.80	5.26	4.66	5.01	5.42	5.34	5.73	6.19	5.77	5.92	5.29	5.75
7	3.91	2.50	2.25	2.65	2.83	2.70	2.71	3.03	3.24	2.33	2.94	3.67	3.68	4.03	3.30	2.97
8	4.57	3.13	2.35	2.62	3.51	3.24	4.22	3.61	3.73	3.94	3.16	4.10	3.48	3.90	4.10	4.00
9	1.84	1.72	2.14	2.50	1.70	1.77	1.54	2.06	.87	2.41	.99	2.02	.51	.92	.66	1.37
10	3.29	2.69	2.53	2.41	2.63	2.43	2.43	2.72	2.55	2.63	2.31	2.63	2.55	2.52	2.59	2.76
11	2.57	1.35	1.30	1.82	1.40	1.92	1.96	1.71	1.40	1.40	1.60	1.89	1.96	1.92	1.92	1.83
12	3.09	3.13	3.51	3.18	3.01	3.17	2.95	3.49	3.72	3.02	3.45	3.59	2.63	3.35	4.17	3.74
13	4.02	3.05	3.27	3.27	2.55	1.81	1.75	2.84	2.46	1.93	0	0	.84	1.72	1.90	0
14	3.91	3.18	3.65	3.23	3.05	3.71	3.44	3.52	3.80	4.26	4.26	4.29	4.23	4.57	3.93	4.29
15	3.55	3.27	2.85	3.21	3.10	3.29	3.13	2.71	3.17	2.71	3.02	3.45	2.89	3.59	3.54	3.78
16	3.90	3.69	3.85	3.69	2.92	3.39	3.38	3.60	4.35	4.06	3.51	2.93	3.59	3.64	3.74	3.40

TABLE 12

Acquisition UCR Magnitudes ($\sqrt{\Delta C}$) for Trace-Unpaired Group

S	TRIALS															
	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16
1	6.74	2.41	2.72	2.88	2.56	2.90	3.12	3.00	2.93	2.85	2.74	3.58	3.09	2.98	3.98	2.93
2	3.19	3.69	3.50	3.26	3.21	2.81	3.18	3.45	3.55	3.51	3.64	3.51	3.64	3.58	3.59	3.72
3	3.55	3.86	3.19	3.00	2.14	2.21	2.57	3.24	3.15	3.18	3.31	3.51	3.86	3.64	3.74	3.28
4	3.10	1.81	2.00	2.03	1.14	1.94	1.62	1.12	1.59	1.59	1.59	1.59	2.00	1.62	1.13	1.62
5	6.84	2.90	2.82	3.23	1.41	1.65	2.77	1.54	2.09	2.86	3.53	1.79	2.45	2.99	2.71	2.55
6	4.38	4.05	3.55	3.45	3.65	1.69	1.19	3.67	3.22	3.60	3.61	3.67	3.81	3.68	3.93	1.73
7	4.06	4.04	4.72	4.57	4.16	3.40	4.00	4.69	4.76	5.87	5.14	4.29	5.70	5.33	4.55	4.60
8	3.59	3.24	3.45	3.47	3.26	2.55	3.19	3.36	3.59	3.54	3.23	3.62	3.48	3.26	3.70	3.59
9	5.57	2.82	2.40	2.88	2.58	2.90	2.82	3.16	3.50	3.13	4.02	3.70	3.95	4.04	3.99	4.21
10	2.49	1.39	.76	1.66	1.30	1.56	1.44	1.59	1.75	1.64	1.37	1.64	1.54	1.69	1.99	1.74
11	4.04	3.70	2.76	3.24	3.03	3.18	3.30	3.35	3.32	3.52	3.29	3.27	3.03	2.91	2.88	3.22
12	2.62	1.94	2.32	2.13	2.07	2.19	2.41	2.06	2.26	2.25	2.64	2.73	2.49	2.53	2.78	2.78
13	2.63	1.85	1.96	1.61	1.26	1.77	1.44	1.66	1.37	1.44	1.35	1.36	1.47	1.49	1.41	1.59
14	4.62	2.19	1.77	1.95	2.07	1.96	1.81	2.90	2.11	1.75	1.75	1.04	2.20	2.19	2.40	2.33
15	1.71	.80	1.62	.77	1.19	1.11	1.20	.87	.89	.88	1.60	.66	1.45	1.15	.66	.94
16	3.10	2.70	2.68	2.33	2.31	2.08	1.86	1.69	2.08	2.55	2.70	.98	2.50	2.92	2.92	3.15

TABLE 13

Mean UCR Magnitudes ($\sqrt{\Delta C}$) For All 64 *S*s on the Pretest Trials (*A*)

	Trace Paired (n=16)	Delayed Paired (n=16)	Trace Unpaired (n=16)	Delayed Unpaired (n=16)
	2.30	2.54	2.64	3.39
	4.01	2.71	3.28	3.90
	2.50	2.84	2.59	2.14
	4.29	3.54	3.42	3.27
	3.83	3.58	3.33	2.87
	1.34	1.90	2.32	4.58
	1.91	2.63	2.32	2.16
	1.60	3.18	2.52	2.54
	2.93	2.98	1.92	1.69
	2.35	4.16	1.43	3.13
	2.40	1.21	2.62	1.22
	1.39	2.14	2.08	2.45
	2.38	1.39	2.41	2.12
	1.65	2.57	2.31	2.21
	2.86	1.11	.85	1.85
	1.81	3.40	2.25	3.57
Overall Means	2.47	2.62	2.39	2.69

TABLE 15

Mean UCR Magnitudes ($\sqrt{\Delta C}$) for All 64 *Ss* on Test Trials (*C*)

	Trace Paired (n=16)	Delayed Paired (n=16)	Trace Unpaired (n=16)	Delayed Unpaired (n=16)
	1.65	3.16	3.42	3.84
	3.52	3.58	3.80	3.31
	1.85	3.72	3.96	2.80
	4.62	2.59	1.44	4.42
	4.32	2.36	3.17	2.55
	3.67	2.52	3.61	5.56
	2.78	3.41	5.08	4.01
	2.09	1.98	3.50	4.11
	2.83	3.21	4.85	1.66
	1.94	4.64	1.55	3.11
	2.13	1.48	3.17	1.79
	2.55	1.92	3.07	4.17
	3.13	1.52	1.42	1.89
	1.68	3.16	2.42	3.35
	3.77	.91	.98	4.01
	2.39	4.34	3.34	4.24
Overall Means	2.87	2.78	3.05	3.43

TABLE 14

Mean UCR Magnitudes ($\sqrt{\Delta C}$) for All 64 Ss on Last 4 Acquisition Trials (B)

	Trace Paired (n=16)	Delayed Paired (n=16)	Trace Unpaired (n=16)	Delayed Unpaired (n=16)
	1.24	2.23	3.25	3.80
	3.13	2.67	3.63	3.58
	3.28	3.03	3.63	2.71
	5.37	2.01	1.59	4.30
	3.58	2.01	2.68	2.60
	2.70	2.29	3.29	5.68
	2.36	2.74	5.04	3.50
	1.17	1.30	3.51	3.87
	1.63	1.47	4.05	.86
	2.02	4.27	1.74	2.60
	1.72	.43	3.01	1.91
	2.16	1.37	2.64	3.47
	2.20	1.00	1.49	1.12
	1.44	3.04	2.28	3.45
	3.20	1.08	1.05	4.34
	1.50	3.00	2.87	3.59
Overall Means	2.42	2.12	2.86	3.21

TABLE 16

Mean UCR Magnitude ($\sqrt{\Delta C}$) Difference Measures Between Pretest Trials (*A*) and Last 4 Acquisition Trials (*B*) For All 64 *Ss* (*A*-*B*)

Trace Paired (n=16)	Delayed Paired (n=16)	Trace Unpaired (n=16)	Delayed Unpaired (n=16)
1.06	.31	-.61	-.41
.88	.04	-.35	.32
-.78	-.19	-1.04	-.57
-1.08	1.53	1.83	-1.03
.25	1.57	.65	.27
-1.36	-.39	-.97	-1.10
-.45	-.11	-2.72	-1.34
.43	1.88	-.99	-1.33
-1.30	1.51	-2.13	.83
.33	-.11	-.31	.53
.68	.78	-.39	-.69
-1.77	.77	-.56	-1.02
.18	.39	.92	1.00
.21	-.47	.03	-1.24
-.34	.03	-.20	-2.49
.31	.40	-.62	-.02

TABLE 17

Mean UCR Magnitude ($\sqrt{\Delta C}$) Difference Measures Between Pretest Trials (A) And Test Trials (C) For All 64 Ss (C-A)

Trace Paired (n=16)	Delayed Paired (n=16)	Trace Unpaired (n=16)	Delayed Unpaired (n=16)
.65	-.62	-.78	-.45
.49	-.87	-.52	.59
.65	-.88	-1.37	-.66
-.33	.95	1.98	-1.15
-.49	1.22	.16	.32
-2.33	-.62	-1.29	-.98
- .87	-.78	-2.76	-1.85
-.49	1.20	-.98	-1.57
.10	-.27	-2.93	.03
.41	-.48	-.12	.02
.27	-.27	-.55	-.57
-1.16	-.22	-.99	-1.72
-.75	-.13	.99	.23
-.03	-.59	-.11	-1.14
-.91	.20	-.13	-2.16
-.58	-.94	-1.09	-.63

TABLE 18

Mean UCR Magnitude ($\sqrt{\Delta C}$) Difference Measures Between Test Trials (*C*) And Last 4 Acquisition Trials (*B*) For All 64 *Ss* (*C*-*B*)

Trace Paired (n=16)	Delayed Paired (n=16)	Trace Unpaired (n=16)	Delayed Unpaired (n=16)
.41	.93	.17	.04
.39	.91	.17	-.27
-1.43	.69	.33	.09
- .75	.58	-.15	.12
.74	.35	.49	-.05
.97	.23	.32	-.12
.42	.67	.04	.51
.92	.68	-.01	.24
1.20	1.74	.80	.80
-.08	1.37	-.19	.51
.41	1.05	.16	-.12
.39	.55	.43	.70
.93	.52	-.07	.77
.24	.12	.14	-.10
.57	-.17	-.07	-.33
.89	1.34	.47	.65

BIOGRAPHICAL SKETCH

Ronald Baxter was born in New York City, New York, on October 13, 1934. He was graduated from Brookline High School in Brookline, Massachusetts in June, 1952. In the Fall of that year he enrolled at Boston University from which he was graduated in June, 1956 with a Bachelor of Arts degree in psychology. In September of 1956 he enrolled at the University of New Mexico where he spent one year doing graduate work in psychology.

After serving six months in the U. S. Army he entered the University of Florida, for graduate studies in psychology. In January, 1961, he received the Master of Arts degree in psychology from this institution. He continued his graduate work at the University of Florida toward the Doctor of Philosophy degree in psychology by completing an internship at the Pittsburgh Child Guidance Center, Pittsburgh, Pennsylvania in August 1962. From that time he remained on the staff of that organization while completing the research requirements for his doctorate.

Ronald Baxter is married to the former Arlene Platten. He is a member of Psi Chi and Sigma Xi.

This dissertation was prepared under the direction of the chairman of the candidate's supervisory committee and has been approved by all members of that committee. It was submitted to the Dean of the College of Arts and Sciences and to the Graduate Council, and was approved as partial fulfillment of the requirements for the degree of Doctor of Philosophy.

December 19, 1964

E. Ruffin Jones
Dean, College of Arts and Sciences

Dean, Graduate School

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